

# Long-term persisting cognitive sequelae of traumatic brain injury and the effect of age

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## Long-Term Persisting Cognitive Sequelae of Traumatic Brain Injury and the Effect of Age

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This study examined the notion that mild to moderate traumatic brain injury (TBI) may have persistent effects that become evident upon neurocognitive testing in a phase in which the effects of physiological aging become manifest. Neurocognitive performance was tested in 25 middle-aged and 20 old subjects who had sustained mild to moderate TBI, on average, several decades earlier. The TBI subjects regarded themselves as normal and healthy. The performance of the TBI subjects was inferior to that of matched healthy controls on all aspects of primary and secondary memory and on the majority of tests used to measure speed of performance. There was no interaction between the effects of TBI and those of age, and the performance of middle-aged TBI subjects was similar to that of old controls. The results are taken to indicate that TBI sustained earlier in life may cause permanent sequelae in specific domains of cognitive functioning and that it might attenuate the age-related decline in cognitive functioning. Most striking, however, was that these deficits were not perceived as a limiting factor in everyday life, which suggests that coping strategies may be important.

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It is well established that mild to moderate traumatic brain injury (TBI) may give rise to the development of postconcussional symptoms, in which both physiogenic and psychogenic influences have an important role (Bohnen and Jolles, 1992; Lishman, 1988). The Hollywood head injury myth, which suggests that the hero can sustain a severe blow to the head but still go on in the next scene without any apparent ill effects, may have contributed to the persisting skepticism about the role of organic factors in the development of a postconcussional syndrome (Evans, 1992). Ample evidence derived from neuropathological (Leestma, 1991), neurophysiological (Hayes et al., 1986; Schoenhuber and Gentilini, 1989), neuroimaging (Levin et al., 1992), postmortem (Oppenheimer, 1968), and neuropsychological studies (Binder, 1986; Bohnen and Jolles, 1992; Green et al., 1992) strongly suggests that organic factors might be involved in postconcussional syndromes. These findings prompted the proposal that the postconcussional syndrome should be recognized as an etiological entity to be classified in DSM-IV (Brown et al., 1994).

In subjects with mild to moderate TBI, postconcussional symptoms may persist well beyond 6 months (Alves et al., 1993), 1 year (Middleboe et al., 1992; Rutherford et al., 1979), or even 3 to 5 years (Edna and Cappelen, 1987), which suggests that the

effects of mild to moderate TBI might not be completely reversible. Levin et al. (1987) and others, however, suggest that a single uncomplicated mild TBI produces no permanent disabling neurobehavioral impairment. Yet several potential risk factors for persisting postconcussional symptoms have been identified, of which lower educational, intellectual, and socioeconomic level, sex, alcohol abuse, repeated TBI, and skull fracture are the most important. Age has also been identified as a possible risk factor for persisting postconcussional symptoms (Edna and Cappelen, 1987; Evans, 1992). A rather alarming neuropsychological follow-up study by Mazzucchi et al. (1992) showed diffuse deterioration in 28%, moderate deterioration in 25%, and dementia in 21% of TBI subjects aged over 50 years, which implies that mild TBI could be followed by severe consequences. Similar, although less dramatic, results were obtained in studies by Houx et al. (1991b; 1993), who found that biological life events (BLE), such as mild TBI, could accentuate the effect of normal biological aging in subjects who considered themselves to be normal and healthy. The usual age-related decline in cognitive functioning, as found in tasks involving new information, planning of new activities, and effortful, controlled processing (Botwinick, 1981; Craik and Byrd, 1982) and the general slowing of behavior (Salthouse, 1985) might become manifest in middle age, when the normal biological aging process becomes evident. The results of a multiple cohort study suggested that BLE might even give rise to predementia

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stages of Alzheimer's disease (Jolles et al., 1993). Houx and co-workers (1991a), however, did not investigate whether the objectively established cognitive deficits in BLE-affected subjects could be correlated to subjective ratings of cognitive functioning. Using the results of a controlled postal survey carried out 1 to 5 years after mild TBI, Bohnen et al. (1994) found that those who had experienced a TBI had many more neurasthenic symptoms than those who had not.

The objective of the current study was first to test the hypothesis that neurocognitive performance is compromised in mild to moderate TBI patients many years after the trauma, even in subjects who do not have complaints. This hypothesis was tested by comparing healthy middle-aged and elderly subjects with mild to moderate TBI in their histories with matched healthy control subjects. The second objective of the study was to evaluate whether there is an interaction between age and TBI. An interaction of age and group in a two-way analysis of variance (ANOVA) design would be indicative of a cumulative effect of age and TBI, whereas group effects without an interaction with age would indicate that mild to moderate TBI in otherwise healthy subjects attenuates the cognitive deterioration caused by age. For this reason, two age groups of TBI subjects were compared. The choice of the neuropsychological tests was based upon earlier studies by Bohnen et al. (1992a) and Houx et al. (1991a, 1991b).

## Methods

### Subjects

Male and female volunteers who had recovered from TBI and who considered themselves normal and healthy were invited to participate in the study by means of an editorial in several local newspapers. Forty-five subjects with mild to moderate uncomplicated TBI were eventually selected from the 110 subjects who initially applied for the study. The remaining subjects did not meet the inclusion criteria. Subjects were preselected over the telephone to ensure that none of them had a medical history of complicated TBI (*i.e.*, bone or skull fractures), a major psychiatric illness known to be characterized by cognitive deficits, previous brain damage caused by stroke or disease, alcohol or drug abuse, impaired vision or hearing, or an insufficient command of Dutch. The time elapsed since the trauma was set at minimally 1 year to include only subjects who were in a stable phase. All participants considered themselves completely recovered. Only subjects who were aged between 40 and 59 years (group I,

middle-aged) or older than 60 years (group II, elderly) were included in the study. Two control groups of 45 volunteers were drawn from a larger population of subjects who were normal and healthy ( $N = 262$ ; Houx et al., 1991a) and free from previous TBI. Control subjects were selected by means of matching to the individual TBI subjects with respect to age ( $\pm 1$  year), sex, and level of education ( $\pm 1$  point). The latter was assessed by a Dutch scoring system (Verhage, 1964) that comprises a scale ranging from 1 (primary education not finished) to 7 (university degree). There were, thus, two TBI groups and two control groups with individually matched pairs. The mean ages of middle-aged and old TBI subjects at testing were 49.4 and 69.8 years, respectively; the mean ages of controls were 48.9 and 70.4 years, respectively. In both the experimental group and the control group, there were 10 male and 15 female middle-aged and 10 male and 10 female old subjects. The mean educational levels of middle-aged and old controls were 4.1 and 3.5, respectively. In TBI subjects, these were 3.9 and 3.1, respectively. These differences were not statistically significant.

The procedure for recruitment of the controls has been described elsewhere (Houx et al., 1991b). In short, they were recruited by means of advertisements placed in local newspapers, and they were paid for their participation. The advertisement stressed that participants should be healthy. All participants underwent the same procedure of neuropsychological evaluation as the experimental group. All subjects were free of significant somatic, neurological, and/or psychiatric disorders; alcohol abuse; TBI; and skull fractures. Depression scores for the controls were not available, but all subjects denied complaints of lowered mood, and they did not report significant problems with memory or concentration. The control subjects can thus be regarded as a representative selection of the middle-aged and old populations (Houx et al., 1991a, 1993).

### Procedure

After informed consent was obtained, the subjects who were eligible for the study answered a postal questionnaire and received a neurocognitive evaluation, which was part of an extensive neuropsychological investigation. The standardized postal questionnaire yielded a subjective report on medical history and cognitive functioning after TBI. Zung's self-rating scale for depression (Zung, 1965) and the 47-item Utrecht Coping List (UCL; Schreurs et al., 1988) were completed, as were a checklist of post-concussive symptoms, which distinguished between

typical postconcussional symptoms and rather aspecific psychovegetative functional symptoms (Bohnen et al., 1992b), and a self-rating scale concerning quality of sleep (Mulder-Hajonides van der Meulen, 1980). Severity of TBI as a possible predictor of outcome was estimated by asking the subjects about coma duration, memory loss, and postconcussive complaints. Coma duration and memory loss were scored on a 4-point scale as less than 15 minutes, 15 minutes to 1 hour, 1 to 24 hours, or more than 24 hours. The duration of postconcussional complaints was also scored on a 4-point scale as being either less than 1 week, 1 to 4 weeks, 1 to 6 months, or 6 months and longer. The subsequent summation of the scores on the three scales yielded a crude measure of the severity of TBI. This estimation of severity is consistent with that of the alternative to the Glasgow Coma Scale suggested by Stambrook, who used posttraumatic amnesia, coma duration, and age as indirect markers of TBI severity in his study of mild to severe TBI patients (Stambrook et al., 1993).

#### *Neurocognitive Tests*

The *Visual Verbal Learning Test* (VVLТ) is a version of the Rey Auditory Verbal Learning Test (Brand and Jolles, 1985; Lezak, 1995). The test consists of a list of 15 monosyllabic and concrete nouns in Dutch, which are presented in five trials on a computer screen. These nouns are frequently used and are learned early in life. Items are presented in the same sequence at a rate of one every 2 seconds. Each trial ends with a free recall of the words (free immediate recall). Twenty minutes after the fifth trial, the subject is requested to recall as many words as possible (free delayed recall). A yes/no recognition test consisting of the 15 former words and 15 new but similar words is given after the delayed recall test. The variables used are number of correctly recalled words on the first trial as a measure of primary memory; the total number of correct words over the five trials as a measure of learning capacity; the number of correct words on delayed free recall as a measure of retrieval from secondary memory; and the number of correct responses on delayed recognition as a measure of memory consolidation.

The *Stroop Color Word Test* (SCWT; Lezak, 1995) is a perceptual interference task that consists of three cards. The test examines the speed at which color names are read (SCWT card I) and the speed at which colored patches are named (SCWT card II). The SCWT card III involves color names again, but the printing ink is different from the color name. The variables are response times for cards I, II, and

III. Interference is expressed by the percentage of extra time needed for card III relative to the average of the first two cards:

$$(III - .5 * [I + II]) / (.5 * [I + II]) * 100\%$$

The *Concept Shifting Test* (CST; Houx and Jolles, 1994) is an adaptation of the Trail-Making Test, which is a test of visual conceptual and visuomotor tracking. The CST consists of three subtasks. In each subtask, the subject has to mark small circles with digits or letters in ascending order on an A4 size sheet of paper. The subject is requested to do this as quickly as possible. On each test sheet, 16 small circles are grouped in a larger circle. In the smaller circles, the test items (numbers [CSTA], letters [CSTB], or both [CSTC]) appear in a fixed random order. The subjects are requested to cross out the items in the right order, in the same way as in the Trail-Making Test. In three identical zero-tasks, all 16 empty circles are to be crossed out clockwise as quickly as possible. The variables are response time in CSTA and CSTB as a measure of visual conceptual and visuomotor tracking and CSTC as an index of concept shifting ability. The relative contribution of the need to shift between two concepts to the total time needed for subtask C (percentage) is conveniently computed with the formula:

$$(CSTC - .5 * [CSTA + CSTB]) / (.5 * [CSTA + CSTB]) * 100\%$$

where CSTA, CSTB, and CSTC denote the total time taken to complete the test part. The average time for the three zero-tasks is used as a measure of basic motor speed.

*Intelligence.* Intelligence was estimated by using the Groningen Intelligentie Test (Groningen Intelligence Test; Luteijn and van der Ploeg, 1982), which is a test of general intelligence in The Netherlands.

#### *Rating Scales*

*Zung's Self-Rating Scale for Depression* (Zung, 1965) was used to measure depression as a clinical entity. The crude total score is expressed as a percentage of the maximum score (80), yielding an index of depression.

The *UCL* (Schreurs et al., 1988) contains 47 items describing seven styles of coping. Factor analytically derived coping styles are active problem-solving, seeking distraction, avoidance and passive expectancy, seeking social support, depressive reaction, expression of emotion and anger, and comforting cognitions.

*Postconcussive Symptoms* (Bohnen et al., 1992b) is a factor analytically derived checklist of postcon-

TABLE 1  
*Characteristics of Middle-Aged and Old Subjects Who Sustained a Mild to Moderate TBI*

	Middle-aged ( <i>N</i> = 25)		Old ( <i>N</i> = 20)		<i>t</i>	<i>p</i>
	Mean	Range	Mean	Range		
Intelligence (GIT)	113.6	(87.0 to 132.0)	115.2	(87.0 to 133.0)	−0.46	NS
Zung's Self-Rating Scale for Depression <sup>a</sup>	49.3	(27.5 to 71.3)	46.0	(32.5 to 66.3)	1.10	NS
Postconcussional symptoms <sup>b</sup>						
PCS-I	24.6	(16.0 to 34.0)	21.9	(14.0 to 32.0)	1.81	NS
PCS-II	21.8	(14.0 to 33.0)	20.9	(15.0 to 33.0)	.62	NS
Sleep questionnaire <sup>c</sup>	61.3	(28.0 to 86.0)	61.4	(42.0 to 83.0)	−.02	NS
Severity of TBI <sup>b</sup>	3.8	(2.0 to 7.0)	3.7	(2.0 to 8.0)	.24	NS
Time elapsed since TBI (yrs)	26.4	(1.0 to 52.0)	35.7	(2.0 to 63.0)	−1.74	NS
Number of TBIs	1.5	(1.0 to 4.0)	1.2	(1.0 to 2.0)	.99	NS
Age at TBI	23.0	(5.0 to 49.0)	34.1	(2.0 to 78.0)	−2.02	< .05

<sup>a</sup>The crude total score is expressed as a percentage of the maximum score (80).

<sup>b</sup>See text for explanation.

<sup>c</sup>Percentage of maximal possible complaints concerning sleep.

cussive symptoms consisting of a factor concerning typical postconcussional symptoms (11 high-loading items; PCS-I), and of a factor concerning rather aspecific psychovegetative functional symptoms (13 high-loading items; PCS-II).

*Subjective sleep quality* (Mulder-Hajonides van der Meulen, 1980) was rated on a 14-item, 5-point scale questionnaire covering aspects of sleep, such as the initiation and maintenance of sleep, sleep interruptions, return to sleep after waking early in the morning, capability to function at peak efficiency upon waking, and the total time the subject reported sleeping at night. The crude total score was expressed as a percentage of the maximum score (70), yielding an index of subjective sleep quality (good sleep quality = 100%).

#### Statistical Analysis

To estimate global age, TBI, or interaction effects, values of VVLT trials 1 through 5, Stroop subtasks I, II, and III, and CST subtasks A, B, and C were used in a repeated-measures ANOVA. The accepted level of significance was set at .05 to indicate tests worth using for further analysis. In subsequent analyses with regard to memory performance, a two-way ANOVA was performed by using the following variables: number of correctly recalled words on the first trial; the total number of correct words over the five trials; the number of correct words on delayed free recall; and the number of correct responses on delayed recognition. For analysis of attentional performance, the time needed for Stroop subtasks I, II, and III and the interference score were used in a two-way ANOVA to analyze differences between TBI subjects and controls. The relative contribution of the need to shift between two concepts to the total time needed for subtask C and

the averaged time of the three zero-tasks were processed in the same way as in the test for memory and attentional performance. The accepted level of significance for all two-way ANOVAs was set at .05. A two-way ANOVA was used to estimate the accuracy with which the various tasks were performed, as reflected by the number of errors, corrections, omissions, and double responses. This analysis did not reveal any significant differences between groups on the neuropsychological tests.

Zung's self-rating scale for depression, the 47-item UCL, the checklist of postconcussive symptoms, and the rating scale for quality of sleep were compared with test performance and injury-related variables by using Pearson's *r*.

## Results

#### Patient Characteristics

Evaluation of the characteristics of the patients in the two TBI groups (Table 1) showed that there were no statistically significant differences between the two groups except for age at the time of TBI: the middle-aged subjects were younger than the old subjects at the time the TBI was sustained ( $t = -2.02$ ,  $df = 43$ ,  $p < .05$ ). There were no differences between middle-aged and old TBI subjects in severity, number of years elapsed since TBI, or number of TBI ( $t = .24$ ,  $df = 43$ , NS;  $t = -1.74$ ,  $df = 43$ , NS; and  $t = .99$ ,  $df = 43$ , NS, respectively). All subjects reported having sustained coma and memory loss, but three middle-aged and seven old subjects reportedly had no postconcussional complaints. The duration of coma, memory loss, and postconcussional complaints was not correlated (Pearson's *r*) with neuropsychological outcome, but the age at which TBI was sustained was positively correlated

TABLE 2  
Psychometric Differences Between Subjects Who Sustained a Mild to Moderate TBI and Controls<sup>a</sup>

Variable	Controls (N = 45)				TBI (N = 45)				TBI effect <sup>a</sup>	Age effect <sup>b</sup>	Interaction effect <sup>b</sup>
	Middle-aged		Old		Middle-aged		Old				
	Mean	SD	Mean	SD	Mean	SD	Mean	SD			
VVLT											
Trial 1	6.2	1.4	5.7	1.4	5.2	1.6	4.5	1.7	< .001	NS	NS
Total in five trials	50.8	6.2	49.0	5.8	44.2	7.5	38.6	9.8	< .001	< .05	NS
Delayed recall	11.4	1.9	11.3	2.3	8.6	3.1	7.1	2.8	< .001	NS	NS
Delayed recognition	14.3	1.0	14.9	0.2	14.0	1.6	13.8	1.6	< .05	NS	NS
SCWT											
SCWT card I	39.0	5.5	45.3	4.3	48.0	9.0	48.7	7.5	< .001	< .05	NS
SCWT card II	52.5	7.7	61.1	8.2	62.1	13.9	62.4	9.1	< .05	< .05	NS
SCWT card III	83.3	13.7	103.8	15.6	98.5	30.4	113.3	22.5	< .01	< .001	NS
SCWT (% slowing) <sup>c</sup>	82.7	22.4	95.9	28.8	77.9	26.8	103.4	24.0	NS	< .001	NS
CST											
CST subtask A	23.3	8.6	25.3	9.6	22.5	6.3	27.6	7.0	NS	< .05	NS
CST subtask B	26.3	9.6	29.0	10.8	27.1	9.0	34.2	14.2	NS	< .05	NS
CST subtask C	37.2	17.3	43.2	23.3	40.2	39.2	43.2	14.1	NS	< .05	NS
CST (% Slowing) <sup>d</sup>	47.1	32.8	53.9	34.8	37.4	25.2	40.8	20.4	NS	NS	NS
Zero-task	5.9	1.4	6.9	2.3	6.9	2.5	8.2	3.3	< .05	< .05	NS

<sup>a</sup>Performance on main test variables. Data for means and SD are expressed as time in seconds, except for the Visual Verbal Learning Test, for which the number of words recalled is shown.

<sup>b</sup>Levels of significance for CHI, age, and interaction effects; NS, not significant.

<sup>c</sup>SCWT interference score is expressed as the percentage of extra time needed for part III, relative to the average for the first two parts:  $(III - .5 * [I + II]) / (.5 * [I + II]) * 100\%$ .

<sup>d</sup>The relative contribution of the need to shift between two concepts to the total time needed for subtask C (percentage) is computed with the formula:  $(CSTC - .5 * [CSTA + CSTB]) / (.5 * [CSTA + CSTB]) * 100\%$ .

with visual conceptual and visuomotor tracking as measured by CST (CSTA,  $r = .32$ ,  $p < .05$  and CSTB,  $r = .41$ ,  $p < .01$ ). Psychometric differences between the main test variables of TBI subjects and controls are summarized in Table 2.

#### Effects on Memory Performance

The effects on memory performance are shown in Figure 1. Primary memory, expressed as the number of correctly recalled words on trial 1, was affected in TBI subjects in the absence of age or interaction effects ( $F[1,86] = 12.19$ ,  $p < .001$ ;  $F[1,86] = 3.27$ , NS; and  $F[1,86] < 1$ , respectively). Measures of secondary memory performance were also affected by TBI. Repeated-measures ANOVAs on trials 1 through 5 showed TBI subjects to perform worse than their matched controls ( $F[1,86] = 29.20$ ,  $p < .001$ ). Although less pronounced, a significant effect of age was also demonstrated in the absence of an interaction effect ( $F[1,86] = 5.56$ ,  $p < .05$  and  $F[4,344] = 1.46$ , NS, respectively). Both TBI and aging seemed to reduce the subjects' capacity to learn new material, as expressed by the five-trial sum score (two-way ANOVA,  $F[1,86] = 29.21$ ,  $p < .001$  and  $F[1,86] = 5.56$ ,  $p < .05$ ). As far as delayed recall after 20 minutes was concerned, TBI subjects showed deficits in active retrieval from memory without age or interaction effects ( $F[1,86] = 41.68$ ,

$p < .001$ ;  $F[1,86] = 2.26$ , NS; and  $F[1,86] = 1.56$ , NS, respectively). TBI subjects also had difficulty in passively retrieving material that had been consolidated into memory ( $F[1,86] = 6.88$ ,  $p < .05$ ). Again, age or interaction effects could not be found ( $F[1,86] < 1$  and  $F[1,86] = 2.95$ , NS, respectively).

#### Effects on Stroop and Concept Shifting Tests

Repeated-measures ANOVA showed that TBI subjects were slower than controls in the SCWT (Figure 2) and that old subjects were slower than middle-aged subjects ( $F[1,86] = 10.24$ ,  $p < .01$  and  $F[1,86] = 11.80$ ,  $p < .001$ ). There was no interaction effect ( $F[2,170] = 1.73$ , NS). Two-way ANOVA showed significant TBI and age effects on all three cards. TBI tended to have a stronger impact than age on highly automated skills such as reading (card I;  $F[1,86] = 17.85$ ,  $p < .001$  and  $F[1,86] = 5.78$ ,  $p < .05$ , respectively). TBI and aging had similar effects on color naming (card II;  $F[1,86] = 6.29$ ,  $p < .05$  and  $F[1,86] = 4.31$ ,  $p < .05$ , respectively). This was not the case for color-word interference (card III), for which performance seemed to be mainly affected by age (TBI,  $F[1,86] = 7.09$ ,  $p < .01$ ; age,  $F[1,86] = 14.57$ ,  $p < .001$ ). When the amount of interference on card III was corrected for the time needed for card I and card II, the effect of TBI disappeared, leaving only an effect of age (TBI,  $F[1,86] < 1$ ; age,  $F[1,86] =$

12.81,  $p < .001$ ). This means that TBI subjects were slower in reading and naming colors but did not show accentuated interference as a result of TBI.

Overall measures of visual conceptual or visuo-motor tracking (CST) revealed effects of age but not of TBI (repeated-measures ANOVA,  $F[1,86] = 4.86$ ,  $p < .05$ ;  $F[1,86] < 1$ , respectively; two-way ANOVAs,  $F[1,86] = 4.36$ ,  $p < .05$ ;  $F[1,86] = 4.59$ ,  $p < .05$ ; and  $F[1,86] = 4.13$ ,  $p < .05$ , respectively). TBI did not affect the performance on the subtasks A, B, and C ( $F[1,86] < 1$ ;  $F[1,86] = 1.66$ , NS; and  $F[1,86] < 1$ , respectively). When the performance on subtask C was related to the time needed for subtask A and subtask B, the effect of age disappeared (age,  $F[1,86] < 1$ ; TBI,  $F[1,86] = 3.45$ , NS; interaction,  $F[1,86] < 1$ ). The basic motor speed seemed to be affected by TBI and age to roughly the same extent (TBI,  $F[1,86] = 5.06$ ,  $p < .05$ ; age  $F[1,86] = 5.20$ ,  $p < .05$ ; interaction,  $F[1,86] < 1$ ).

#### Effects on Psychological Variables

Zung's self-rating scale for depression was positively correlated only with the scores for SCWT cards I and II ( $r = .47$ ,  $p < .01$ ;  $r = .41$ ,  $p < .01$ , respectively). According to normative data derived from a normal, healthy population (Zung and Durham, 1973), the TBI subjects in our study could not be characterized as suffering from major depression. The TBI subjects who, according to the Zung scores, had a somewhat depressed mood, had a lower perceived quality of sleep ( $r = .55$ ,  $p < .001$ ), more PCS-I and PCS-II symptoms, and a more depressive reaction pattern, as measured by the UCL ( $r = .74$ ,  $p < .001$ ;  $r = .72$ ,  $p < .001$ ;  $r = .65$ ,  $p < .001$ , respectively). In the TBI subjects, intelligence tended to be negatively correlated to self-rating of depression ( $r = -.36$ ,  $p < .01$ ). Coping styles could not be consistently correlated to neuropsychological outcome. Active problem solving, as expressed by the UCL, tended to be positively correlated with intelligence ( $r = .41$ ,  $p < .01$ ) and negatively correlated with sleep and PCS-I and PCS-II symptoms ( $r = -.39$ ,  $p < .01$ ,  $r = -.40$ ,  $p < .05$ ;  $r = -.43$ ,  $p < .01$ , respectively). Depressive reaction patterns correlated with complaints concerning sleep ( $r = .35$ ,  $p < .05$ ) but correlated more strongly with PCS-I and PCS-II symptoms ( $r = .62$  and  $.59$ ,  $p < .001$ ).

#### Discussion

One major question of the present study was whether middle-aged or old subjects who are considered to have recovered from mild to moderate TBI could be characterized by a decreased neurocognitive performance compared with that of

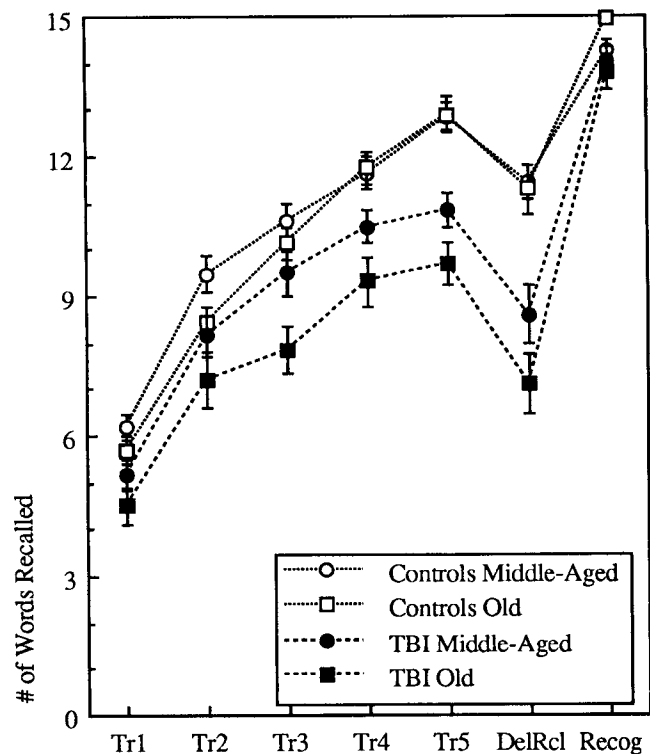


FIG. 1. Mean performance on Trials 1 through 5, Delayed Recall (DelRcl) and Recognition (Recog) in the Visual Verbal Learning Test, of middle-aged subjects with a mild to moderate TBI versus optimally healthy age peers, contrasted to the performance of old subjects affected by TBI versus optimally healthy age peers (vertical bars denote Standard Errors of the Mean).

matched controls. The period between trauma and neuropsychological evaluation in this study extended well beyond the traditional period of recovery. A second major question was whether there was evidence that TBI accelerated cognitive aging. The results show that the performance of the TBI subjects was significantly inferior to that of the matched controls in all tests of primary and secondary memory used. An effect of age could be demonstrated on the learning capacity but not on delayed recall. Not only did TBI subjects do less well than controls in consolidating material into memory, but they also showed a marked difficulty in passively retrieving this information from memory. These data are new in that, until now, there were no studies in which neurocognitive performance was evaluated so long after the TBI.

The most striking finding of our study is that although the TBI subjects had reportedly made a good recovery and did not have any cognitive complaints at the time of selection for the study, they still performed worse than their matched controls. We found no robust correlation between indices of the severity of TBI and neuropsychological outcome, al-

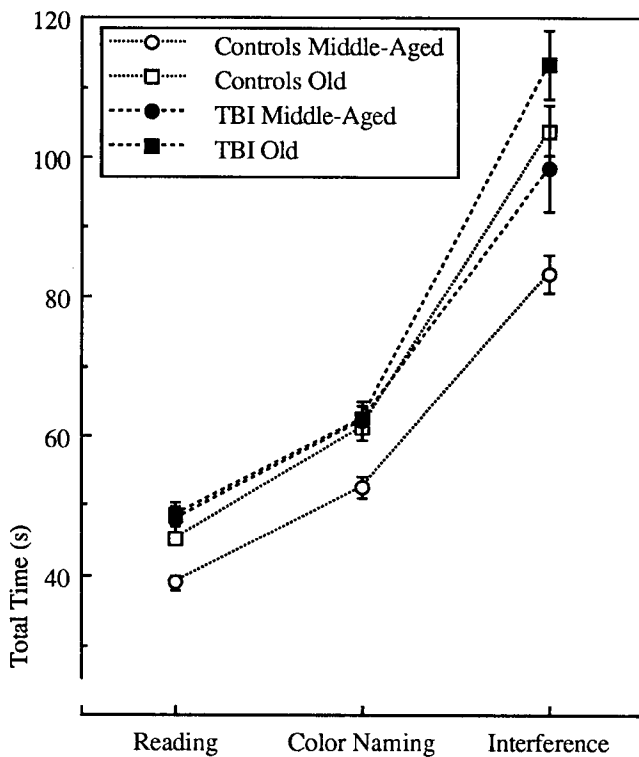


FIG. 2. Mean performance on three cards of the Stroop Color Word Test, of middle-aged versus old subjects affected by a mild to moderate TBI, contrasted to the performance of their optimally healthy age peers (vertical bars denote Standard Errors of the Mean).

though the age at which TBI was sustained turned out to be poorly but positively correlated with visual conceptual and visuomotor tracking scores. This suggests that cognitive sequelae might be relatively independent of the severity of the TBI in our population, which, on average, sustained TBI 3 decades earlier. It is possible that after recovery, the subjects reached a steady state beyond which their cognitive deficit did not improve. The pattern of the scores of the middle-aged TBI subjects on the SCWT was very similar to the pattern of the old controls (Figure 2). The older TBI subjects, however, did not perform disproportionately worse than their younger peers. We consider that these scoring patterns are indicative of accelerated cognitive aging caused by the effect of TBI. In the middle-aged TBI subjects, the usual age-related decline in cognitive functioning seemed to occur earlier than in their peers not affected by TBI. As such, the data obtained in the present study provide important information with respect to the relation between cognitive aging and age-extrinsic factors, such as mild to moderate TBI. The present results strengthen the notion proposed by Houx et al. (1991a) and Jolles et al. (1993) that minor health-related factors (so-called BLE) are very

important in determining the age at which the physiological aging process leads to cognitive deterioration. It must be emphasized, however, that our population might not be a truly representative sample of the general TBI population, because our subjects represent a group of TBI subjects that voluntarily responded to newspaper advertisements. Moreover, they had to regard themselves as normal and healthy, thus excluding subjects with possibly more serious sequelae of previous TBI. More research is needed to deepen our insight into the relation between TBI and age.

As far as the nature of the effects on neurocognitive performance are concerned, our findings are similar to those of Levin et al. (1988), who found that approximately one fourth of TBI subjects with relatively preserved intellectual functioning manifested defective memory in both auditory and pictorial measures of memory when they were tested 5 to 15 and/or 16 to 42 months after injury. These results should be considered with caution because the patients in Levin et al.'s group represent a sample of moderate to severe TBI. Both moderate and severe TBI groups, however, still showed deficits in memory performance 16 to 42 months after injury. Gupta and Ghai (1991) found TBI subjects to perform significantly poorer on both immediate and delayed free recall. Hall and Bornstein (1991) found that the poorer total recall scores of subjects with mild TBI could not be related to the duration between injury and neuropsychological evaluation and suggested that this poorer performance was a lasting feature of memory function after such an injury. The fact that in the present study, coma duration, memory loss, and duration of postconcussional complaints could not in any way be correlated with the neuropsychological outcome partly supports the findings of Strugar et al. (1993), who concluded that subjects with mild TBI do not necessarily have to have been unconscious to have memory deficits.

As far as perceptual interference is concerned, Posner and Snyder (1975, p. 57) concluded from SCWT studies that color naming and reading go on in parallel and without interference until close to the output: "If they result in look-up of the same name, the overall reaction time is speeded; if they produce different names and a vocal output is required, the word tends to compete with the color name and reaction time is increased." Our TBI subjects needed significantly more time than the middle-aged controls for all cards of the SCWT, as did the older control subjects. With respect to interference (SCWT card III), no interaction of age with TBI could be demonstrated. When corrected for the basic speed for cards I and II, only age seemed to be



a relevant factor in explaining the extra slowing with card III. This suggests that the TBI subjects in this study did not have a selective attention deficit but that they did have a more generalized reduced rate of information processing, thus confirming the results of a study by Stuss et al. (1985). Stuss and colleagues state that residual brain damage secondary to TBI might be expressed by an impaired information processing capacity. Reeder and Logue (1994) stressed the lack of processing resources in explaining memory deficits in their study and even concluded that a possible paucity in attentional capacity cannot be held responsible for subsequent memory deficits. The results seem to support the ideas of Symonds (1962), who found it questionable whether even mild TBI would ever be completely reversible.

Although information processing seemed to be the main cognitive process affected in the TBI subjects, the literature provides some evidence that there are deficits in perceptual and motor processes (Van Zomeren et al., 1984). This would be compatible with the lower basal CST motor speed of TBI subjects in our study. Visual conceptual and visuomotor tracking, reflected by the CST score, could not, however, distinguish between the two groups.

The fact that the TBI subjects did not complain about their cognitive limitations might be attributed in part to the role of coping strategies. Our middle-aged subjects showed a somewhat more active attitude, *i.e.*, they tended to seek more distraction and social support than the old TBI subjects did. The latter difference proved to be statistically significant. The subjects with a less active coping style tended to have more complaints concerning sleep, as well as PCS-I and PCS-II symptoms. We might further question whether intelligence also plays a role in determining complaints after TBI. According to Sternberg (1982), intelligence enables the subject to acquire, process, classify, and integrate information obtained from the environment. This might mean that the more intelligent a person is, the better he or she will be able to deal with cognitive challenges in everyday life. In our study, the more intelligent subjects showed lower self-ratings of depression and a stronger tendency to active problem-solving. A lower level of intelligence might, in fact, accelerate the decline in brain reserve capacity associated with aging and, thus, might give rise to an earlier onset of clinical symptoms (Satz, 1993). Our TBI subjects had an average intelligence score that was somewhat higher than that of the general population in The Netherlands. This fact might have contributed to the observed lack of long-term neuroathenic symptoms in our TBI subjects, although

this possibility can only be verified by longitudinal studies.

An explanation for the discrepancy between neuropsychological outcome and cognitive complaints could be that we used self-ratings for coma duration, posttraumatic amnesia, and duration of complaints after TBI rather than the stricter Glasgow Coma Scale. Because posttraumatic amnesia obscures the recall of facts related to injury, TBI subjects usually are not able to give a reliable report, and, therefore, they compare themselves with how they were before the trauma. We might, however, question whether recall in mild to moderate TBI is as strongly affected as it is in severe TBI. Another factor that possibly contributes to the discrepancy is the fact that subjective reports on events that took place a long time ago tend to be rather unreliable regardless of the possible effects of posttraumatic amnesia.

### Conclusions

In conclusion, this study shows that although subjects who had sustained a TBI many years previously might be characterized by objective cognitive deficits or even by an attenuated cognitive aging, they do not necessarily have cognitive complaints. This lack might be attributed to the fact that they sustained a TBI many years ago. This might mean that over the years, they learned to cope with cognitive limitations in everyday life. The fact that our subjects were, on average, more intelligent might have contributed, in part, to this. Our findings suggest that future research should aim at determining the relative contribution of personal resources, such as the premorbid level of intelligence, mental stability, and family support, and the extent to which these resources can compensate for the decline in brain reserve capacity associated with aging.

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